1971 - 1972

REPORT NO. 88
JANUARY 1973

CENTER FOR DISEASE CONTROL

INFLUENZA - RESPIRATORY DISEASE SURVEILLANCE

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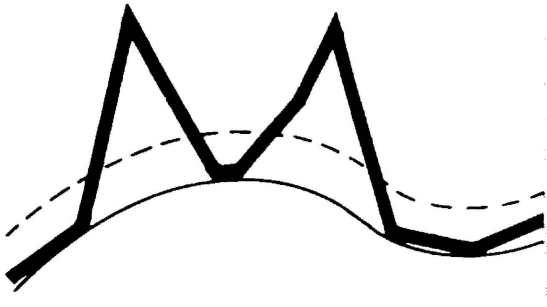

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE

PREFACE

Summarized in this report is information received from State Health Departments and other pertinent sources, domestic and foreign. Some of the information is preliminary. It is intended primarily for the use of those with responsibility for disease control activities. Anyone desiring to quote this report should contact the original investigator for confirmation and interpretation.

Contributions to the surveillance report are most welcome. Please address to:

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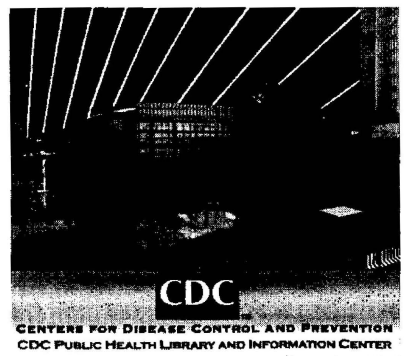

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DHEW Publication No. (HSM) 72-8084

INFLUENZA SURVEILLANCE 1971-72

INTRODUCTION

Influenza occurs each year in the United States. While it is not a reportable disease, states are requested to report outbreaks to the Center for Disease Control. Approximately 25 states currently report cases of influenza or influenza-like disease in their own regularly published morbidity reports. In addition to these reports from the states, CDC conducts periodic telephone surveys of state epidemiologists throughout the influenza season. We also receive mortality data weekly from 122 cities throughout the United States and laboratory data from over 60 laboratories throughout the nation. These sources and their respective data are summarized in this report for the 1971-1972 influenza season.

I. SURVEILLANCE SUMMARY

A) General Data

Through periodic telephone surveys to state epidemiologists and health officials, specific information was obtained regarding the presence of influenza, its location, extent, and severity. Although rigorously accurate statistical data were generally not available, this type of information does give a broad overall picture of the spread of the disease.

The 1971-1972 influenza season was not prefaced by known widespread seeding throughout the country in the summer and fall months; in fact, only 1 documentation of influenza was received by the Viral Diseases Branch, CDC, in the summer of 1971. Scattered reports of influenza-like illness were received in the first 2 weeks of November, and the first telephone survey was undertaken on November 17-18, 1971. At that time, no confirmation of respiratory illness due to influenza was obtained.

The first confirmed influenza outbreak occurred in Connecticut in early December 1971, and this report was followed by documentations from New Jersey and Utah. A telephone survey undertaken December 21, 1971, revealed scattered documented isolated outbreaks across the country in Connecticut, Kansas, Michigan, New Jersey, and Utah; influenza-like disease was reported from 11 additional states (Figure 1).

On January 3, 1972, when a telephone survey was undertaken, widespread disease* was noted only in Connecticut, New Jersey, and Rhode Island, while regional outbreaks were observed in Michigan and Wisconsin. Isolated outbreaks of respiratory disease due to influenzavirus were observed in the following states: Colorado, Kansas, Illinois, Iowa, Maine, New York, Ohio, Oregon, South Dakota, Texas, and Utah. Moreover, reports of influenza-like illness were received in 20 additional states (Figure 2).

A telephone survey undertaken on January 17, 1972, showed widespread disease in the District of Columbia, New York City, and in 8 states: Colorado, Connecticut, Delaware, Maine, Massachusetts, Nebraska, New Jersey, and Rhode Island. There were

*The CDC classifies the extent of influenza in 4 categories: 1) isolated cases, 2) isolated outbreaks, 3) regional involvement (outbreaks recognized in contiguous counties, but altogether involving counties comprising less than one-half of the state's population), and 4) widespread involvement (more than half of the counties or more than half of the population).

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regional outbreaks in Idaho, Iowa, Kansas, Louisiana, Maryland, Michigan, Minnesota, Montana, New York, North Carolina, Ohio, Oregon, South Dakota, Texas, and Wisconsin. Isolated outbreaks were observed in 14 states and febrile upper respiratory illness was noted in 11 additional states (Figure 3).

Figure 1
INFLUENZA A_2, 1971-1972

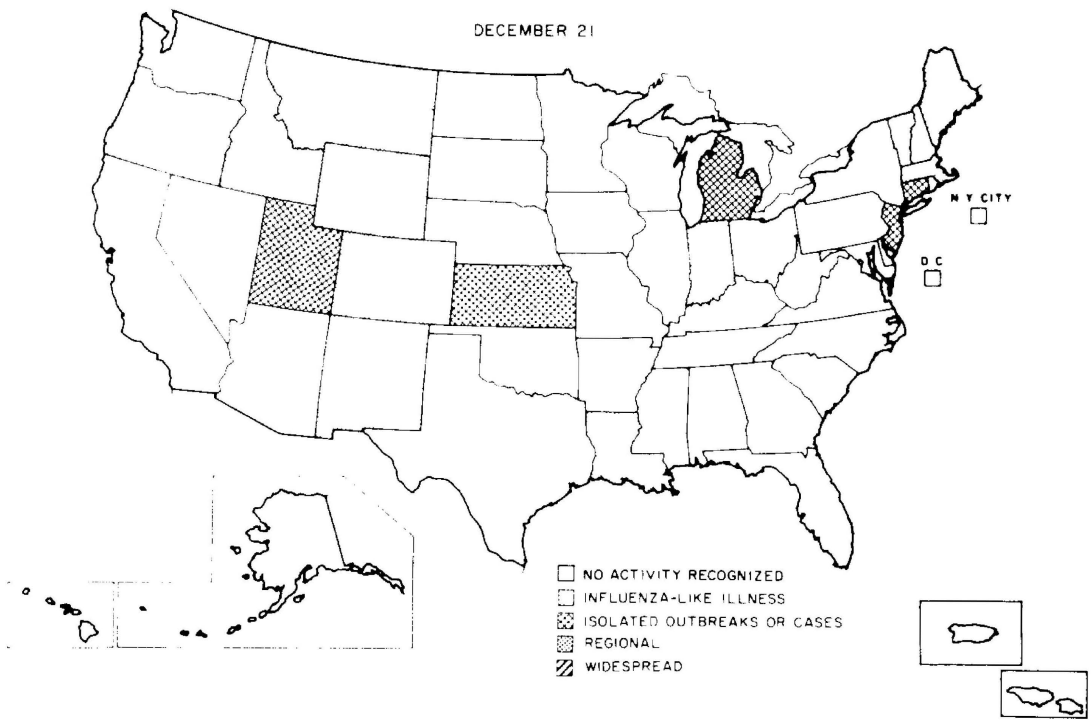


Figure 2
INFLUENZA A_2, 1971-1972

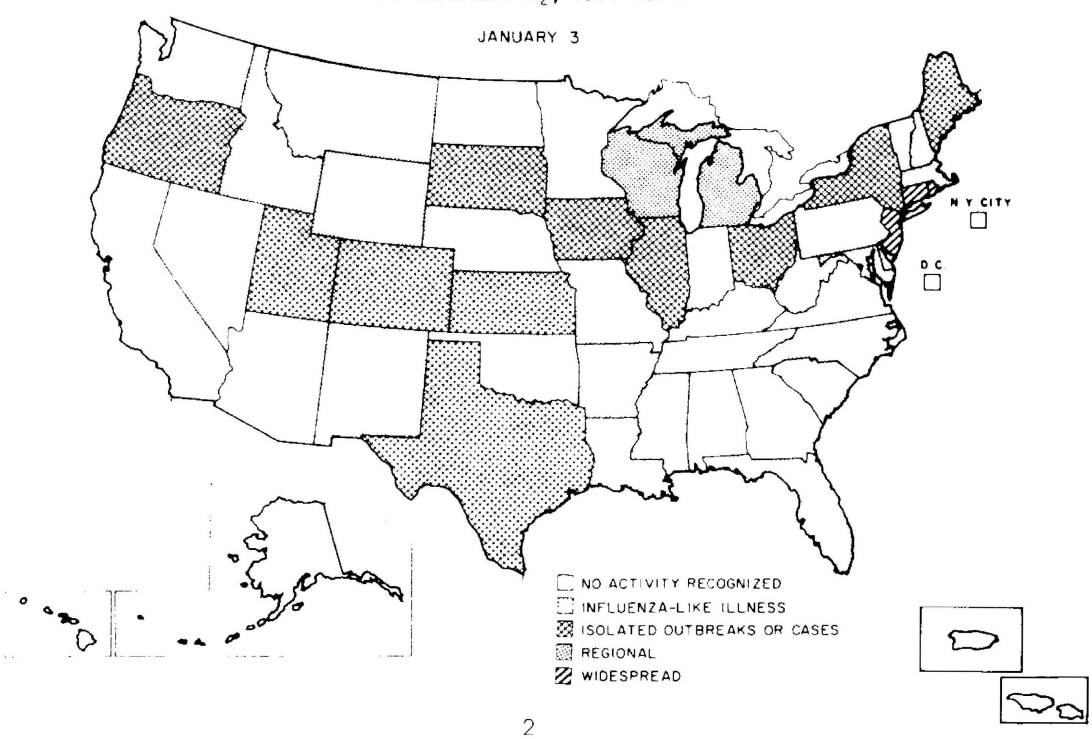
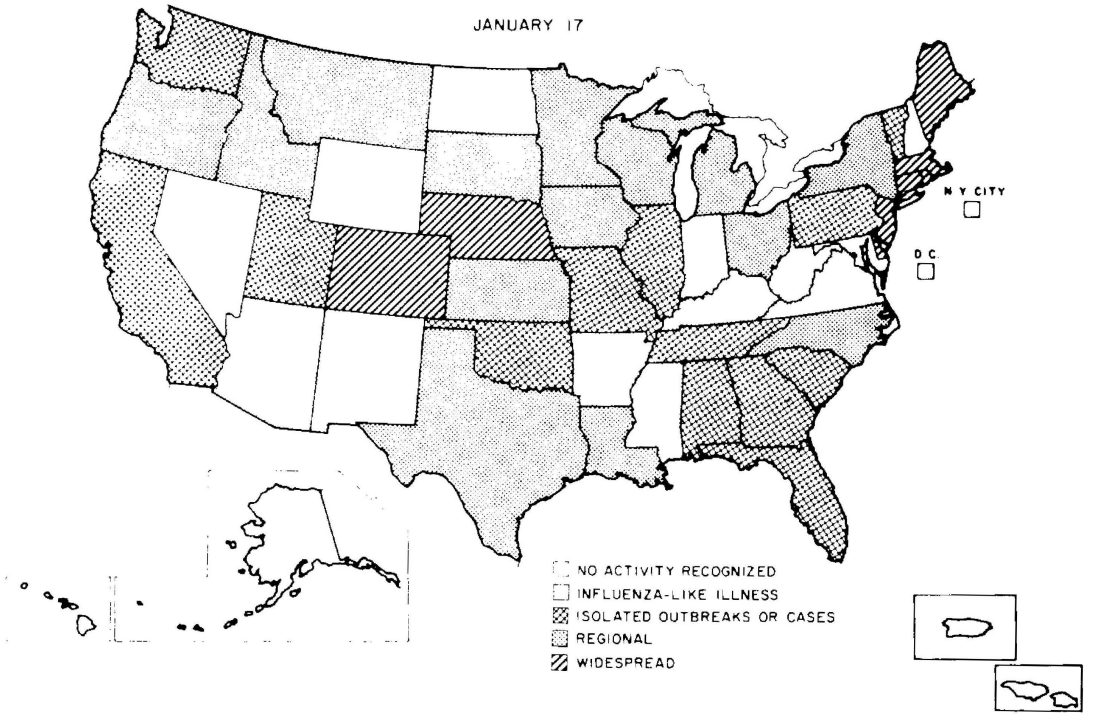


Figure 3
INFLUENZA A_2, 1971-1972



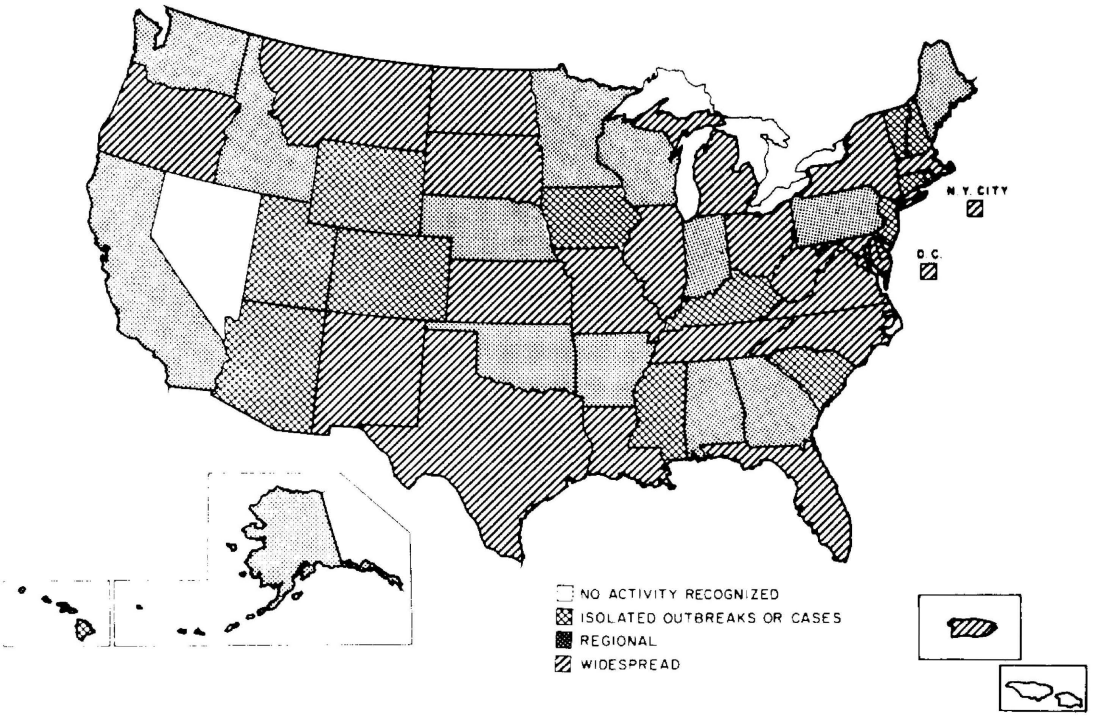
By January 31, widespread disease was noted in 21 states, the District of Columbia, New York City, and Puerto Rico. States involved were Delaware, Florida, Kansas, Illinois, Louisiana, Maryland, Massachusetts, Michigan, Missouri, Montana, New Mexico, New York, North Carolina, North Dakota, Ohio, Oregon, South Dakota, Tennessee, Texas, Virginia, and West Virginia. Regional outbreaks of disease were reported from 14 states, and 14 states reported isolated outbreaks. Colorado, Connecticut, Delaware, Maine, Nebraska, New Jersey, Rhode Island, and Utah all reported decreases in the incidence of influenza at this time (Figure 4).

B) Mortality Statistics

1. Description. Deaths are reported each week from the Vital Statistics Offices of 122 United States cities and are recorded in Table IV of the Morbidity and Mortality Weekly Report (MMWR). They are by place of occurrence of death, thus including deaths of persons whose residence may be elsewhere and not including deaths of residents which occur in other vital statistics jurisdictions. The report is a count of death certificates filed, and each week the deaths recorded include some which may have occurred in preceding weeks. The number of delayed certificates usually increases during holiday periods, causing a drop in the number of deaths reported for the holiday week, followed by an increase when the delayed certificates are included in the report in the succeeding weeks.

This information reflects influenza activity by a rise in mortality usually 2-4 weeks after the clinical disease is noted to be widespread. These data provide the best available epidemiologic evidence of the extent and severity of an epidemic in the country as a whole.

Figure 4
INFLUENZA A_2, 1971-1972



2. Expected Mortality and the Epidemic Threshold. Expected mortality is determined by the use of data for prior years to predict the weekly mortality level for the coming year.[1,2,3] The method works well in general because the same seasonal pattern is observed each year, the same peaks are observed in years without much influenza activity, and the same nadirs are observed almost every year. The exception to this observation is that over a period of years there is sometimes a general upward or downward trend in mortality. Except for infant mortality, this trend has not been very great in recent years.

The expected mortality level is determined by using weekly data for the previous 4- or 5-year period, omitting data for epidemic periods, and fitting the data to the following model by least squares:

$$\hat{y} = u + rt + A_1 \cos\frac{2\pi t}{52} + B_1 \sin\frac{2\pi t}{52} + A_2 \cos\frac{4\pi t}{52} + B_2 \sin\frac{4\pi t}{52}$$

References

1. Collins SD, Lehmann J: Excess deaths from influenza and pneumonia and from important chronic diseases during epidemic periods, 1918-51, Public Health Monogr 10 (PHS Publication 213), US Government Printing Office, Washington, DC, 1953

2. Serfling RE: Methods for current statistical analysis of excess pneumonia-influenza deaths. Public Health Rep 78:494-506, 1963

3. Serfling RE: The current mortality chart. Morbidity and Mortality Weekly Rep 14(1):8-11, 1965

The expected level is obtained by inserting the appropriate value of t in the equation where t is the number of the week from the beginning of the data which was fitted to the model. This procedure allows for a general mean, a slope, and annual and semi-annual cycles in the data, and omission of epidemic data prevents an inflation of the expected level during the influenza season. Except for resulting in a slightly smoother curve and yielding a standard error which forms the basis of the epidemic threshold and the scale on which the graphs are drawn, the procedure is almost equivalent to averaging the deaths for corresponding weeks over the curve-fitting period and using the average as the expected for the next year.

The error mean square for each curve is obtained by summing the squares of the differences in observed and expected values over the curve-fitting period, omitting the data during epidemic periods, and dividing by the appropriate degrees of freedom. The square root of this is the standard error of the curve fit and is the basis for the epidemic threshold, defined as 1.65 standard errors above the expected. Experience has shown that the deviations between observed and expected values are normally distributed in most instances. Thus, the probability that one observation will exceed the threshold is .05, and the probability that two successive ones will exceed the threshold is approximately $(.05)^2$.

3. Construction of the Charts. The reported numbers of deaths are shown as dots joined by line segments. The solid line for each mortality category is the expected number of deaths. The dashed line is the "epidemic threshold", a criterion for recognition of significant deviations in excess of the expected number.

The charts are drawn to a scale that allows the distance between the expected and threshold levels to be constant for every curve. This device allows one to compare the influenza activity between regions by glancing at the regional chart. Although the vertical labels are different, comparison of the absolute distance on the chart between observed and threshold levels between regions shows whether the mortality is significantly higher in one region than another. This is accomplished by allowing 0.3 inches on the original full size chart to represent 1.65 standard errors of measurement for each graph that is drawn.

4. Discussion. To make as accurate a prediction of expected mortality levels as possible, the curve-fitting procedure is repeated each year utilizing the most current full calendar year of data available and dropping the earliest year used previously. This naturally results in a solution to the above equation which is slightly different from the solution for the previous year.

For the present, Flint, Michigan, has been omitted from the list of cities due to difficulties in obtaining the data. Simultaneously, Las Vegas, Nevada, has been added to the Mountain Region. This change in the data base may affect the expected values slightly, as does refitting the curves each year.

Reported by: Statistical Services Activity, Epidemiology Program, Center for Disease Control.

5. Mortality in 122 Cities for the 1971-1972 Influenza Season. Mortality in 122 cities due to all causes for the 1971-1972 influenza season is shown in Figure 5. Pneumonia and influenza mortality for these cities was significantly elevated above the epidemic threshold from the 2nd through the 7th week of 1972. Although influenza was reported throughout the country, the excess mortality was primarily limited to the New England, the East North Central, the Middle Atlantic, and South Atlantic Regions (Figure 6). Excess mortality from pneumonia and influenza for this period was 1900 -- greater than the excess mortality reported in the 1970 influenza epidemic.

C) Comment

Nearly all the influenza A isolates were closely related to A/Hong Kong/8/68 (H3N2) by the hemagglutination inhibition tests. There were a few isolated outbreaks of influenza B.

Although 49 to 50 states reported influenza for 1971-1972, attack rates and school absenteeism were relatively low in many areas. In most parts of the country, the clinical illness was reported to be less severe than in previous Hong Kong influenza epidemics, although the excess mortality was the highest since the introduction of the Hong Kong virus into the United States in 1968-1969 (Figure 7).

Figure 5

MORTALITY IN 122 UNITED STATES CITIES

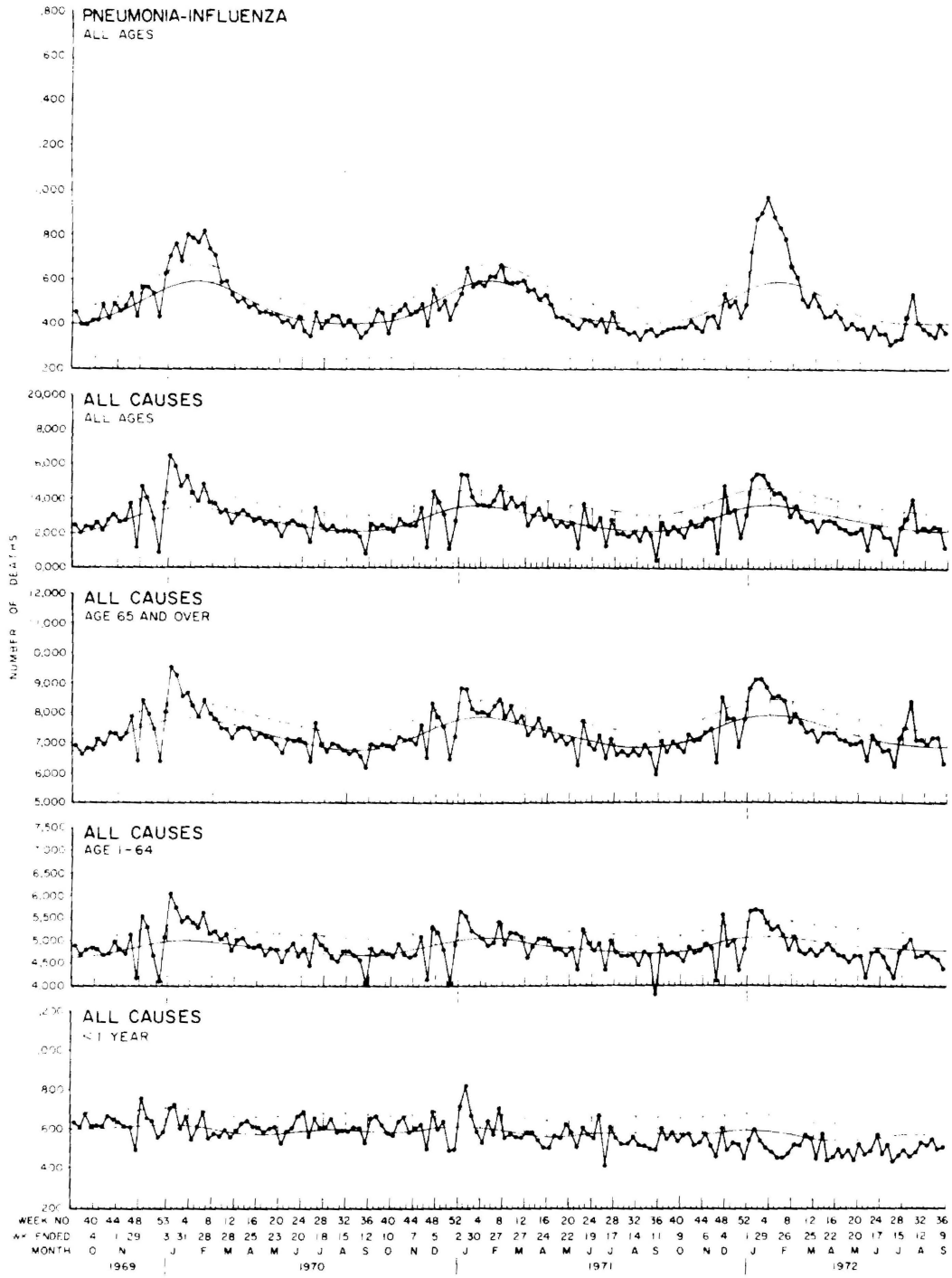


Figure 6

PNEUMONIA-INFLUENZA DEATHS IN 122 UNITED STATES CITIES

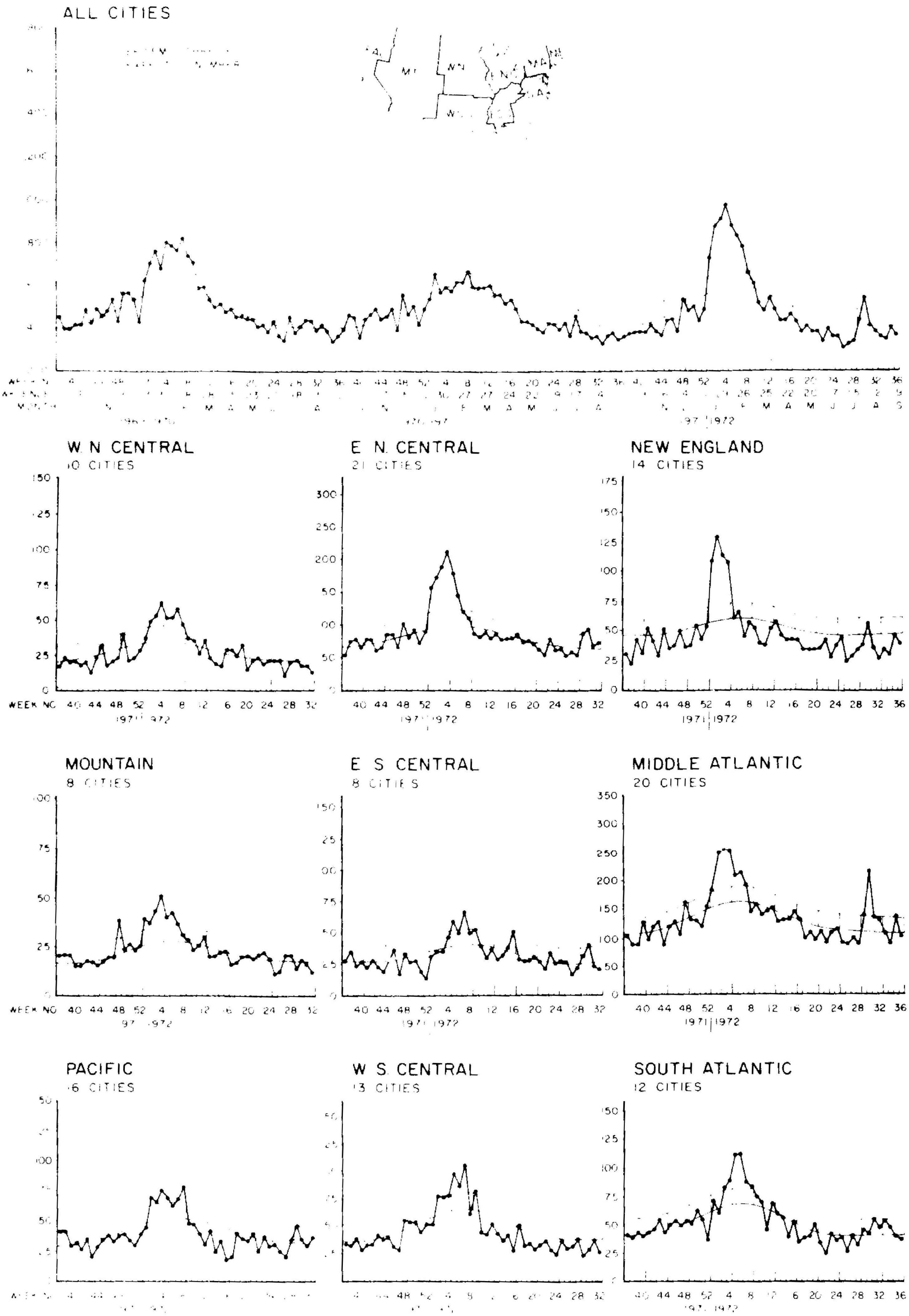
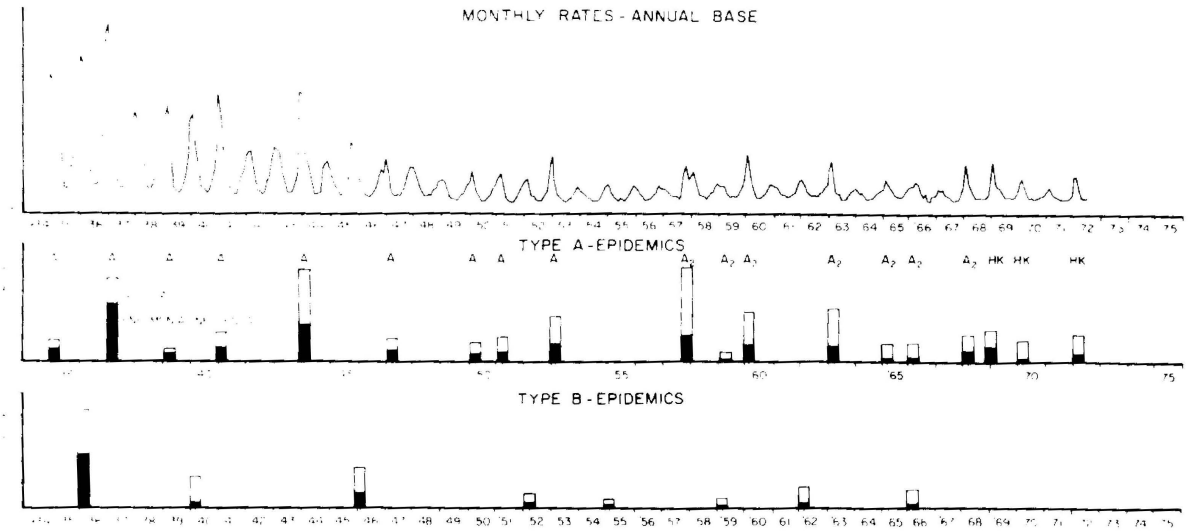


Figure 7

PNEUMONIA-INFLUENZA DEATH RATES BY MONTH
AND
EXCESS MORTALITY DURING EPIDEMIC PERIODS
UNITED STATES, 1934-1972*



*FROM EPIDEMIOLOGY PROGRAM, CENTER FOR DISEASE CONTROL

II. REPORT FROM THE STATES

A) Connecticut

In the United States, the first confirmed case of influenza for the 1971-1972 season occurred in Connecticut. On December 7, 1971, 473 (25%) of the 17,060 students at a high school in Meriden, Connecticut, were absent from school with a febrile illness; 23 teachers were also absent. The illness was characterized by low-grade fever, headache, and sore throat. Less frequent symptoms include cough, earache, and abdominal pain. Specimens were obtained from 8 students, and 3 were positive for the A/Hong Kong strain of influenza. Connecticut went on to have an influenza season characterized by widespread disease.

Reported by: James C. Hart, M.D., State Epidemiologist.

B) New Jersey

The State Department of Health used the traditional tool of excess absenteeism as the chief surveillance mechanism in mapping influenza activity throughout the state.

The first reports of influenza activity came from Passaic County in mid-December. The first virologic as well as serologic confirmations were obtained from cases in Passaic County and in Sussex County just prior to Christmas time. The antigenic characteristics of the influenzavirus recovered from all the patients in 1971-1972 were similar to the prototype Hong Kong strain. Subsequent to the laboratory confirmation in northern New Jersey, additional isolates as well as antibody rises were obtained from patients from throughout New Jersey.

The outbreak appeared to reach its peak in the 1st 2 weeks of January, at least to the extent that disease activity was reflected in school absenteeism. In the last 2 weeks of January, the number of schools with excessive absenteeism dropped significantly.

At the end of January there were still pockets of influenza activity scattered throughout the state but no evidence of widespread disease. In general, the outbreak seemed to start in northern New Jersey and gradually moved southward.

The clinical manifestations of influenza appeared to be milder than in 1968. However, there were at least 6 deaths virologically confirmed as having been caused by influenza.

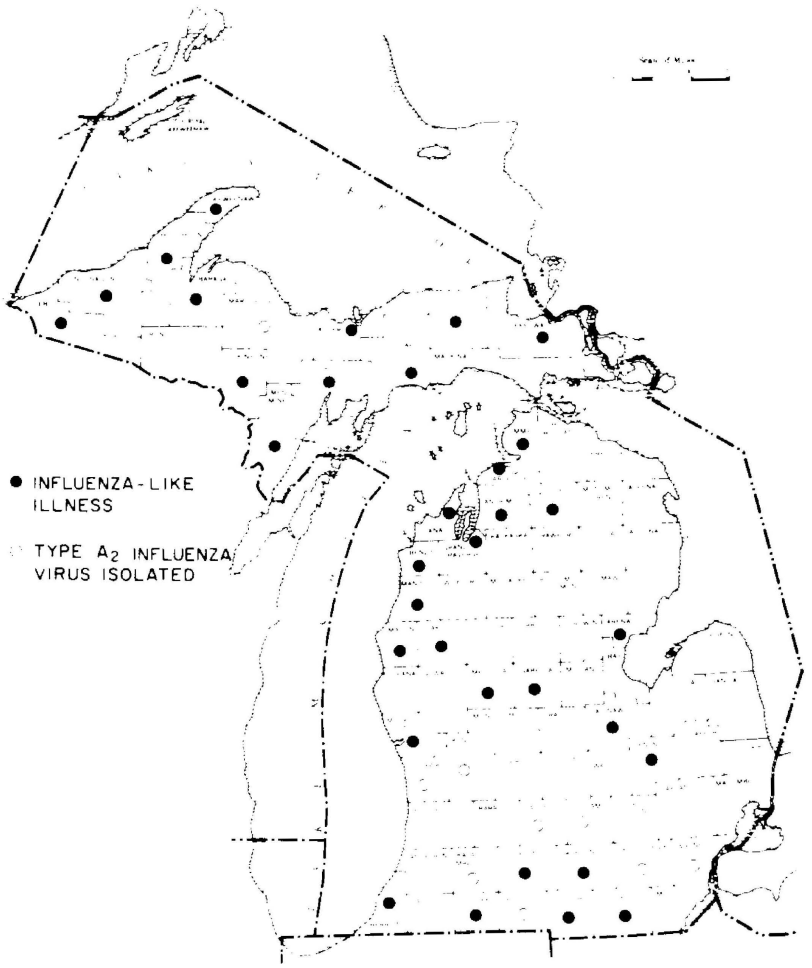
Reported by: Ronald Altman, M.D., Director, Epidemiologic Services, New Jersey State Department of Health.

C) Michigan

In 1971-72, Michigan experienced widespread outbreaks of influenza. Virus was isolated in 9 counties and all viral isolations appeared to be similar to the prototype Hong Kong strain. An additional 34 counties reported influenza-like illness (Figure 8).

Reported by: Norman S. Hayner, M.D., State Epidemiologist.

Fig. 8 INFLUENZA-LIKE ACTIVITY, MICHIGAN, DEC. 6, 1971 - JAN. 14, 1972



D) Florida

Florida had a widespread outbreak of influenza in 1971-72. In this outbreak, the 10th reported case of influenza viremia occurred. The case report follows: Since the recovery of influenzavirus from extra-pulmonary tissue of man and animals was first demonstrated in 1957, the occurrence of viremia in naturally occurring infections has been suspected. Viremia in naturally infected individuals has previously been reported in only 9 cases. The following report describes the isolation of influenzavirus from post-mortem blood of a patient who died in the 1971-1972 influenza outbreak in Florida.

A 59-year-old white female had been in good health until January 1, 1972. Twenty years previously she had undergone left pneumonectomy because of active tuberculosis. Since that time she had had a chronic productive cough, however, from January 1-4 the productive cough became more pronounced and dyspnea developed. She was seen at a local hospital in Florida on January 4 where the diagnosis of active tuberculosis was suspected. Since no isolation facilities were available in the hospital, she was not admitted. The following morning a concerned neighbor visited her and found her dead in a chair in her house. The body was transported to a local hospital where an autopsy was done by the county medical examiner 6 hours later. Microscopic examination of the right lung revealed extensive areas in which the aveoli contained pink staining edema-like material, moderate numbers of acute inflammatory cells, and smaller numbers of lymphocytes and macrophages. No hyaline membranes, interstitial pneumonitis, viral inclusion bodies, or giant cells were present.

A pulmonary vein blood sample was obtained, and serum was stored at -20°C for 5 days before it was sent to the virus laboratory. On the receipt of the serum, a portion was diluted to 10^{-1}. Hank's basic salt solution and both the undiluted and 10^{-1} dilutions were inoculated into replicate tubes of rhesus primary monkey kidney cell cultures from which the growth medium had been decanted. Absorption was allowed to proceed for 1 hour at 37°C after which 1.5 ml of Melnick's B maintenance media containing 5% fetal calf serum was added. Cultures were incubated at 37° and observed daily for cytopathogenic effect (CPE). On day 5, cell sheets in all inoculated tubes showed 2+ CPE. Hemadsorption inhibition was attempted using influenza A, influenza B, and parainfluenza 3 antisera. Inhibition was evidenced by the influenza A antiserum only. These studies and viral isolation were confirmed at CDC.

In all naturally occurring cases of influenza in which viremia has been reported, disease has been severe, requiring intensive care during extended hospitalizations or, has been fatal. These observations appear to indicate a relationship between severity of disease and late viremia.

Reported by: F. M. Wellings, Sc.D., J. J. Shinner, M.D., A. L. Lewis, D.V.M., and C. J. Seabury, R.N., from the Epidemiology Research Center, Bureau of Research, Division of Health, State of Florida Department of Health and Rehabilitative Services, and Pinellas County Medical Examiner, St. Petersburg, Florida.

E) Washington, D.C.

Several indices were used for influenza surveillance in Washington last year. These indices were combined with a "virus watch" (performed by the Virus Laboratory at Children's Hospital) and showed that the most sensitive indicators of an ongoing outbreak of influenza were the number of patients seen in either pediatric screening clinic or the emergency room of a general hospital.

The widespread outbreak in Washington, D.C., was characterized by no significant increase in industrial absenteeism and only a slight increase in school absenteeism. (Table 1 and Figure 3). The prevalent virus was the prototype Hong Kong strain.

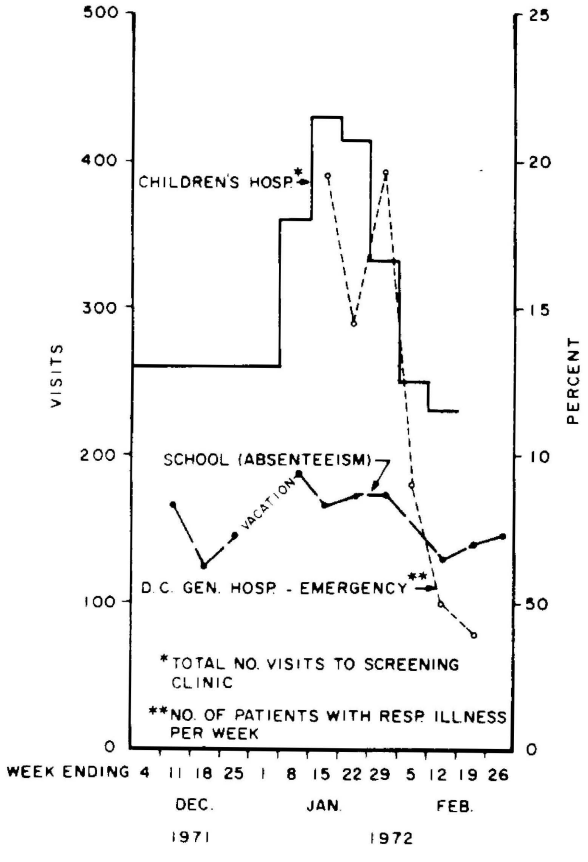
Reported by: Virus Laboratory, Children's Hospital, William E. Long, M.D., State Epidemiologist, and Robert E. Marier, M.D., EIS Officer.

Table 1

Pneumonia Deaths and Influenza Isolates -- Washington, D.C., 1971-72

	Week Ending										
	12/16	12/23	12/30	1/6	1/13	1/20	1/27	2/3	2/10	2/17	2/24
Pneumonia deaths	5	5	2	8	7	6	7	13	7	15	8
Virus isolates (A2)	1	0	6	10	10	15	17	5	2	3	1

Fig. 9 **INFLUENZA SURVEILLANCE, WASHINGTON, D.C., 1971-1972**



F) Iowa

On Wednesday, January 12, 1972, a news item in the Des Moines Register reported the sudden increase of respiratory infections in the Cherokee-LeMars area with school absenteeism running as high as 16%. That afternoon a virologist of the Iowa State Hygienic Laboratory was in LeMars collecting specimens under the direction of local physicians for virologic and serologic examination. On the following day specimens were collected from patients in Cherokee. Convalescent blood specimens were collected from these volunteers on January 27 and 28, 1972.

Throat washings and acute blood specimens were collected from patients who were asked to volunteer for this study. In each case, permission was obtained from the parents before any specimens were collected. The parents were asked to consult their family physicians before making their decision. The local physicians and parents cooperated completely and were very enthusiastic in the project.

The number of students absent from school because of illness in the month of January was compiled for each school in the 2 school systems. With the total enrollment of each school known, the daily absenteeism rate was calculated (Figures 10 and 11). These graphs show that in the Cherokee school system, all of the schools were affected with high attack rates and absenteeism rates peaking above 25% except in Washington High School which was about 16%. There was a rapid buildup in the number of students absent and also a rapid decline within an interval of about 2 weeks from the beginning of the outbreak. In the Cherokee schools, the epidemic peaked on January 12 or 13, 1972.

The course of the influenza outbreak in LeMars was quite different from that seen in Cherokee. First, the attack rate was lower with a peak of less than 9% absenteeism in the junior high school and ranged between 13% and 18% in the elementary grades. Furthermore, at Franklin Elementary School the daily absenteeism rate was between 10% and 12% for nearly 3 weeks. And finally, the daily absenteeism rate for the senior high school was never above 6%, although confirmed cases of influenza were detected in this student population.

Influenzavirus resembling the A/Hong Kong/1/68 (H3N2) strain was isolated from all 4 throat washings collected from students in LeMars and from 4 out of 8 specimens collected in Cherokee. Influenzavirus was also isolated from a throat washing collected by a practicing physician of LeMars. The virus was easily isolated in embryonated eggs.

Fig. 10 DAILY ABSENTEEISM-CHEROKEE, IOWA

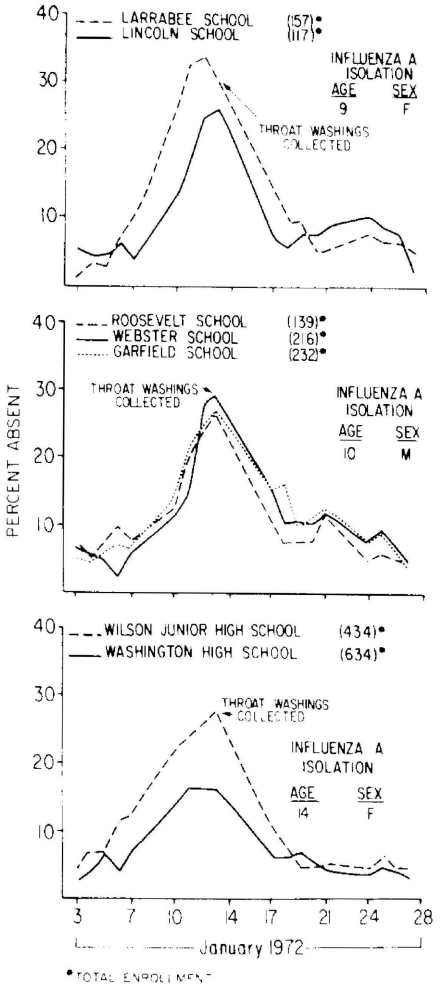
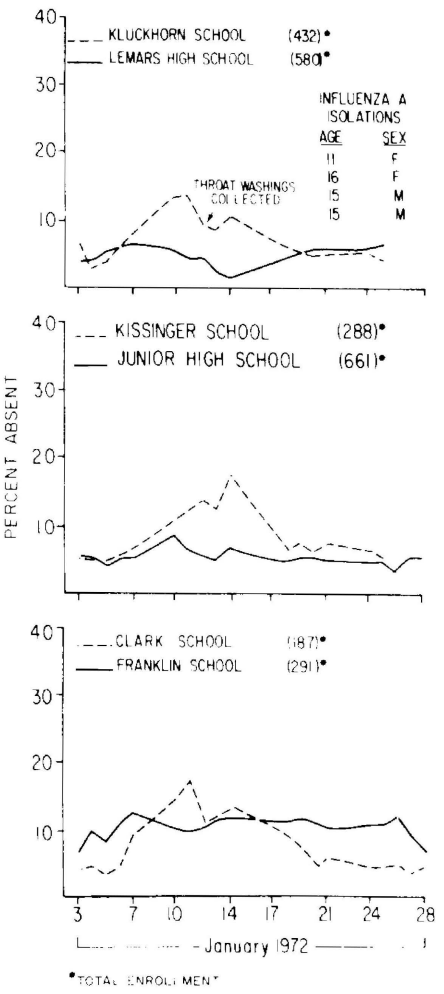


Fig. 11 DAILY ABSENTEEISM- LEMARS, IOWA



All 11 paired blood samples showed diagnostic rises in complement fixation titers for influenza A with titers less than 1:8 in every case for influenza B. These results indicate that the patients were recently infected with influenza A virus. Three single convalescent sera also showed antibody for influenza A and not for B. Antibodies for adenovirus and Mycoplasma pneumoniae were also detected in some of the sera.

Laboratory results clearly indicated that the patients studied were infected with the A/Hong Kong influenzavirus. The rise in antibody titer for influenza A is compatible with a recent infection with this virus. The cultural characteristic of the viruses isolated during these outbreaks are similar to the other A/Hong Kong strains that have been isolated in the laboratory.

There was one viral pneumonia death in Cherokee during the outbreak. This was a 52-year-old man who died about 8 days after onset of illness. Specimens collected shortly before death showed a 1:256 or greater titer for influenza A and less than 1:8 for influenza B, adenovirus and Mycoplasma pneumoniae.

Results of the investigation indicated that the recent high incidence of respiratory illnesses in the Cherokee and LeMars school was caused by the A/Hong Kong influenzavirus.

Reported by: A. M. Reeves, M.D., Commissioner of Public Health, S. L. Hendricks, M.D., Preventive Medical Services, and the State Hygienic Laboratory.

G) Tennessee

From December 1971 to February 1972, outbreaks of influenza-like illness occurred in a few rural counties of Tennessee. Several school systems closed because of high absentee rates. Influenza A2 virus was isolated from patients in areas of the outbreaks and also from residents of Memphis, Nashville, and Knoxville. Statewide weekly official morbidity reports had the greatest totals since 1968.

In order to improve surveillance of influenza in urban areas, the Tennessee Department of Public Health requested reporting of absenteeism from selected schools and factories. Schools in Nashville, Knoxville, Chattanooga, Jackson, and Murfreesboro and industrial plants in Jackson and Chattanooga reported daily absenteeism to their county health department which relayed them to the State Health Department.

Absentee data obtained from Jackson were typical of those from all the reporting cities. They were monitored from November 15 through February. The absentee data from both schools and industrial plants remained stable while the totals of reported influenza cases fluctuated.

These data support the impression of the Department that the amount of true morbidity outside of some rural counties was not as remarkable as official reports suggested.

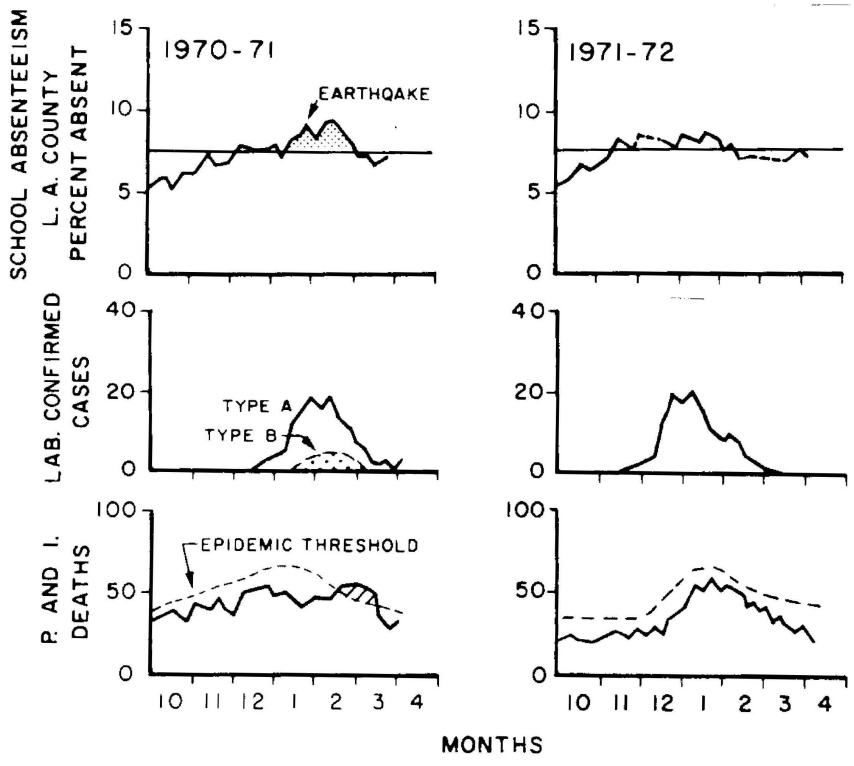
Reported by: R. H. Hutcheson, Jr., M.D., M.P.H., Assistant Commissioner and State Epidemiologist, Tennessee Department of Public Health, and an EIS Officer.

H) California

No major outbreak of influenza was observed in the 1971-1972 influenza season. Pneumonia-influenza deaths were not above the epidemic threshold. The weekly reports of school absenteeism submitted by the 15 counties participating in a California influenza surveillance program indicated that influenza activity was more extensive in many southern areas than in the rest of the state. The influenzaviruses isolated in California were antigentically similar to the A/Hong Kong/8/68 (H3N2) strain (Figure 12).

Reported by: Bureau of Communicable Disease Control, State Department of Public Health.

Fig. 12 INFLUENZA SURVEILLANCE, CALIFORNIA



I) Oregon

Of roughly 400 persons for whom either throat washings for virus isolation or paired sera for serologic specimens were received, 93 persons were either serolog cally confirmed or confirmed by virus isolation, or by both tests, as having infl A2 similar antigenically to the A2 strain formerly designated as A2 Hong Kong 196 Laboratory confirmations were in patients ill with influenza-like symptoms betwee the dates of December 8, 1971, and March 6, 1972. The time of positive findings 7 confirmations for December, 69 for January 1972, 16 for February, and 1 for Mar 1972. In all, 20 of Oregon's 36 counties had at least 1 laboratory confirmed cas A2 Hong Kong influenza for the period. No other strains of influenza were confir by virus isolation for the period, and in only 1 case was there a serologic titer to influenza B antigens. Other respiratory viruses identified in the period were isolation of parainfluenzavirus Type I (in early December) and 1 isolation of para fluenzavirus Type III (in late January). Twelve specimens gave serologic evidence mycoplasma infections during the period. There were comparatively few cases in wh both virus isolation and paired sera were submitted; however, this occurred often enough to indicate that the serologic and virus isolation data were congruent and confirmatory of each other. Influenza A2 was confirmed in every area of the state adequate specimens were taken and the distribution of positive findings was such a confirm the occurrence of a statewide outbreak of A2 influenza.

An effort was made to obtain absenteeism reports from several representative a (Portland, Washington County, Lincoln County, Jackson County, and Lane County). Th data obtained were useful in following the course of the outbreak. Although isolat reports were received of school absenteeism exceeding 50% in a few schools, such hi rates were a rare event and did not occur in any of the schools or industries chose for absenteeism reports. Absenteeism had already peaked in a few areas (such as Ba and Union Counties in far eastern Oregon) when the surveillance system was inaugura (week of December 18, 1971). Absenteeism in State Health Division personnel main office in Portland was also used to supplement Portland Metropolitan Area data.

Jackson County (selected schools) absenteeism peaked during the week ending Jan uary 29th with a 14.4% absenteeism rate for the week (almost exactly double the ave rate of absenteeism for the period). Lane County data were based on adult absentee ism (one large industry and the university student body). The absenteeism rates we much lower (varying from 0.2% to 0.57%), with peak absenteeism being reached in the week of January 8, 1972. Lincoln County absenteeism (based on selected elementary and secondary schools) varied from 4.7% to 15.3% with the peak level of 15.3% reach in the week ending January 29, 1972. The Multnomah County absenteeism (based on se lected elementary and secondary schools) ranged from a low of 1.5% to a peak of 11. for the week ending January 29, 1972. Information from Umatilla and Washington cou ties was too scanty to be useful. The absenteeism among State Health Division per- sonnel (all adults) varied from 3.67% to 16.0%. The peak values of 16.0% were reac on the week ending January 22nd with almost as high a level for the week ending Jan uary 8th (15.3%). This population represents Multnomah, Washington, and Clackamas counties, plus a few persons from Marion County.

The surveillance data served to indicate that this epidemic, although of wide distribution, was of lower intensity than the similar influenza epidemic of 1968-69

Reported by: John A. Googins, M.D., State Epidemiologist.

III. INFLUENZA SURVEILLANCE SUMMARY -- THE WORLD

The influenza season in Europe started in mid-September 1971, in Rumania. The disease was epidemic in other parts of Eastern Europe by early fall, with reports from Bulgaria, Czechoslovakia, Hungary, and Poland. The disease appeared to spread rather slowly, frequently appeared to be clinically mild, and in most instances was due to a Hong Kong virus which was similar to the prototype strain A/Hong Kong/8/68

Influenza spread from Eastern Europe into Austria, but cases were limited pri- marily to the Vienna area, and the clinical disease was relatively mild.

By the end of November, widespread influenza outbreaks were reported in Scandinavia. The clinical illness experience in these outbreaks was described as being relatively mild.

Influenza appeared in Germany in mid-January throughout the country and widespread outbreaks occurred in the Lower Saxony Province. There was also limited activity in France. England had a widespread but mild outbreak of influenza with almost all the cases caused by a virus similar to A/Hong Kong/1/68 (H3N2). In February 1972, the World Influenza Center in London reported that hemagglutination inhibition tests had shown that one of the strains isolated in England during the epidemic, A/England/42/72, presented significant antigenic differences from the prototype A/Hong Kong/1/68 and from the variants A/England/878/69 and A/Hong Kong/107/71.

Influenza-like illness was first noted in Russia in the latter part of November and reached epidemic proportions in late November and early December. The disease was widespread throughout the country.

Influenza was also reported from Ceylon, Lebanon, Switzerland, Algeria, Belgium, Morocco, Grenada, Israel, and Malta.

Although most viruses isolated were basically similar to the prototype strain, A/Hong Kong/8/68, there were 8 strains which were more closely related to the A/Hong Kong/107/71 variant. These were isolated in Hong Kong (2), Germany, Hungary (4), and Algeria. There were also some strains that were more closely related to the A/England/878/69.

In India, there was widespread activity in southern India (Coonoor) from July to November 1971. Seven of the 8 strains isolated in Coonoor were closer to A/England/42/72 than to the prototype A/Hong Kong/1/68.

In the spring of 1972 there were widespread outbreaks of influenza in Korea, Malaysia, and Singapore. The predominant virus isolated was closely related to A/England/42/72. All these areas had widespread influenza in 1971 caused by virus similar to the prototype A/Hong Kong/68.

South Africa (Johannesburg area) and Argentina (Buenos Aires) also had widespread outbreaks in the spring and early summer of 1972 but the virus was similar to the prototype Hong Kong virus.

In Hong Kong in August there were 425 cases of influenza reported to the Hong Kong Health Department with 6 deaths. Most of the viral isolates were type A. The Hong Kong Health Department stated that the number of cases of influenza for August was typical for that month and did not reflect any increased activity.

In Australia there were several outbreaks of influenza. Melbourne and Sydney had moderate outbreaks of disease, however, strains from Australia were similar to A/England/42/72 rather than to the prototype Hong Kong strain. Two outbreaks occurred in military recruits, 1 in Melbourne with an attack rate of 20%, and 1 in Leeuwin with an attack rate of 50%. Influenza A was the etiologic agent in both outbreaks. Western Australia reported widespread disease caused by both influenza A and influenza B.

There were outbreaks of influenza aboard U. S. Naval vessels in the South Pacific. Attack rates were variable but some were as high as 36%. Aboard 1 ship, 15% of those vaccinated less than 6 months previously became ill. The virus isolated was similar to A/England.

In September, widespread outbreaks were reported from Guam, Yap, and the Fiji Islands. The predominant virus has been similar to A/England.

Reported by: The World Health Organization Weekly Epidemiologic Record, Vol 46 and 47, 1971 and 1972, and from the Bureau of Preventive Medicine, U. S. Navy.

IV. LABORATORY REPORT -- WHO INTERNATIONAL INFLUENZA CENTER FOR THE AMERICAS

A) Antigenic analysis of influenzavirus isolates 1971-72

From July 1971 to July 1972 the International Influenza Center for the Americas (IICA) examined the antigenic characteristics of 429 influenza A and 6 influenza B strains from 60 laboratories collaborating in the Influenza Program of the World Health Organization.

Three influenza B strains were received from Trinidad and 3 from Guam. Thes strains closely resemble the type B (B/Victoria/98926/70) viruses which were iso in 1970-71. No influenza B activity was noted in other areas.

In keeping with the new system of nomenclature[1], all influenza A strains were with component-specific hemagglutinin (H) and neuraminidase (N) reference antiser well as with antisera to current strains. The H3 antiserum was prepared in chick against electrophoretically isolated hemagglutinins from the recombinant virus A/ 2/68(H3)-Bel/43(N1). This is the same antiserum which was distributed to collabo laboratories last November. The N2 antiserum was prepared in rabbits against ele phoretically isolated N2 from recombinant virus A/NWS/33(HO)-HK/8/68(N2). This a serum was available upon request to collaborating laboratories.

Nearly all of the 429 influenza A isolates submitted to the Center were closel related to A/Hong Kong/8/68(H3N2) by HI tests. The HI reactions of those few isol which showed some drift away from Hong Kong/68 are given in Table 2.

Table 2

Hemagglutination inhibition (HI) reactions of type A influenzaviruses, 1971-72

	Antisera*			
Antigens	A/Aichi/2/68 (H3)**	A/HK/107/71 (H3N2)	A/Chiba/71 (H3N2)	A/Hawaii/9/72 (H3N2)
A/Hong Kong/8/68(H3N2)	4217+	188	320	320
A/Hong Kong/107/71	320	320	113	226
A/Chiba/71	1780	95	640	80
A/Hungary/2/71	1780	188	113	320
A/Hungary/153/71	1280	188	226	226
A/San Diego/2/72	1490	95	160	113
A/Louisiana/5/72	1780	113	320	226
A/Great Lakes/2/72	1280	95	226	113
A/Mayo Clinic/32/72	890	95	113	80
A/Hawaii/9/72	750	363	160	453

*Chicken antisera treated with receptor destroying enzyme
**Antiserum produced against electrophoretically isolated A/Aichi/2/68 hemagglutinin
+Geometric mean titers of 2 or more HI tests

Thirty strains were selected for analysis by neuraminidase inhibition (NI) tests The NI_{50} titers of the reference serum with the unknown viruses were compared to the serum titer with the reference [A/equine/Prague/1/56(Heq1)-HK/16/68(N2)] strain (Tab 3). All strains tested were clearly of the N2 neuraminidase subtype. However, if differences in NI titer ≥ 0.5 $\log_{10}$ ($\geq$threefold) are considered to be significant, th majority of strains were antigenically different from the reference A/Hong Kong (N2) (Table 3). The neuraminidase antigens of only 4 strains were closely related to A/ Hong Kong/8/68(N2). Strains with variant neuraminidase antigens were not necessaril those with the variant hemagglutinin antigens. This is not unexpected. The two sur- face antigens are known to vary independently.

Although minor changes in the H3 or N2 antigens are reported here among influenz A strains from several geographic areas, the importance of these changes with respec to disease is unknown. None of the strains, however, have been uniquely associated with epidemic influenza.

Reference

1. WHO Committee 1971. A revised system of nomenclature for influenza viruses. Bull WHO 45:119-124

Table 3

Differences between neuraminidase inhibition (NI) titers of 1971-72 influenza A viruses and reference virus A/equine/Prague/1/56(Heq1)-HK/16/68(N2) with rabbit antiserum against isolated Hong Kong/68(N2) neuraminidase

Negative $\log_{10}$ difference from anti HK/68(N2) homologous* serum titer			
<0.5	0.6 - 0.9	1.0 - 1.5	1.6 - 2.0
A/Great Lakes/1/71	A/New Jersey/3/71	A/Florida/1/72	A/Chile/379/71
A/Sao Paulo/1/71	A/Canada/1/72	A/Great Lakes/2/72	A/Hungary/153/71
A/Albany/1/72	A/Colorado/7/72	A/Hawaii/1/72	A/Mayo Clinic/32/72
A/North Dakota/3/72	A/Columbia Univ./2/72	A/Hawaii/4/72	A/Mayo Clinic/52/72
	A/England/42/72	A/Iowa/2/72	
	A/Georgia/24/72	A/Mayo Clinic/11/72	
	A/Hawaii/5/72	A/Mayo Clinic/15/72	
	A/Hawaii/9/72	A/Memphis/3/72	
	A/Hungary/2/72	A/Minnesota/1/72	
	A/San Diego/2/72	A/Oklahoma/14/72	
		A/Oregon/6/71	
		A/Puerto Rico/3/72	

*1:350,000

The pattern of antigenic drift seen to date in the hemagglutinin of the H3 strains is not unlike that seen with the Asian strains 4 years after the 1957 pandemic. Antigenic drift among the Asian strains was gradual for about the first 6-7 years. It does not necessarily follow that the antigenic behavior of the Hong Kong virus will continue to parallel that of the Asian virus. First, the neuraminidase of the 1957 virus was new, and the neuraminidase of the 1968 virus was not. Second, the human population has increased and conditions of virus transmission and spread may have been altered.

The small number of hemagglutinin variants detected in 1971-1972 is thought to be a reasonable representation of the actual proportion of circulating strains. This assessment of the frequency of antigenically different strains is probably more accurate than it may have been in the past since larger numbers of strains were examined in the IICA and in the World Influenza Center in London. The large number of strains were made available through the active support of the WHO program by an increasing number of collaborating laboratories. In the next few years the virus can be expected to exhibit even greater antigenic drift. It then becomes more important to monitor antigenic changes in influenzaviruses isolated from wide geographic areas.

B) Influenzavirus Nomenclature

A revised system of nomenclature for the influenzaviruses was adopted on January 1, 1972, on recommendation of a study group of the World Health Organization[2]. The new system was intended to correct 2 major inadequacies: 1) the previous influenza A subtype designation did not take into account both the neuraminidase and hemagglutinin antigens and, therefore, did not fully describe the antigenic character of the virus; and 2) there was no provision for expressing antigenic relationship among viruses isolated from different animal species.

The new nomenclature consists of a strain designation and a description of the hemagglutinin and neuraminidase antigens. The strain designation contains 1) a

Reference

2. Ibid.

description of the antigenic type of ribonucleoprotein (A, B, or C); 2) the original host species (unless human); 3) geographic origin; 4) strain number; 5) year of isolation. The antigenic description for influenza A strains includes in parentheses an index describing the antigenic character of the hemagglutinin subtype and an index describing the antigenic character of the neuraminidase subtype.

Table 4

Revised System of Nomenclature for Influenzaviruses

Antigenic subtypes of hemagglutinin and neuraminidase of influenza A viruses of human origin

H subtype	Reference strain	N subtype	Reference strain
H0	A/PR/8/34 (H0N1) A/Weiss/43 (H0N1)	N1	A/PR/8/34 (H0N1) A/FM/1/47 (H1N1)
H1	A/FM/1/47 (H1N1) A/England/1/51 (H1N1) A/Denver/1/57 (H1N1)		
H2	A/Singapore/1/57 (H2N2) A/England/12/64 (H2N2) A/Tokyo/3/67 (H2N2)	N2	A/Singapore/1/57 (H2N2) A/Hong Kong/1/68 (H3N2)
H3	A/Hong Kong/1/68 (H3N2)		

RECOMMENDATION OF THE PUBLIC HEALTH SERVICE ADVISORY COMMITTEE ON IMMUNIZATION PRACTICES

INFLUENZA VACCINE

INTRODUCTION

Influenza occurs in the United States every year, but the incidence and geographic extent vary widely. Periodically, it appears in epidemic form as a result of antigenic variation in prevalent viruses and the relative susceptibility of the population. Both type A and type B influenzaviruses undergo antigen changes. Such changes usually occur slowly, but occasionally they are rapid and and abrupt. Epidemics caused by type A influenzaviruses occur more frequently and are generally more severe than those caused by type B.

The effectiveness of inactivated influenza vaccines* has been variable, and protection has been relatively brief. This has contributed to recommendations only for selective use in persons at high-risk. Vaccine for 1972-73 has more antigen than prior products and should give better results. It should be given to chronically ill patients and possibly to older persons in general. These two groups appear to be more vulnerable than others to serious cases of influenza and its complications. Because some influenza occurs each year, annual immunization of "high-risk" patients is indicated as a routine procedure regardless of the amount of influenza expected in any specific geographic area.

INFLUENZAVIRUS VACCINES

The Division of Biologics Standards reviews influenza vaccine formulation regularly and recommends reformulation, when indicated, to include contemporary antigens. The influenza vaccine this year is different from that available in 1971-72. Although the type A strain present in 1971-72 is retained, its potency has been increased from 400 to 700 chick cell agglutinating (CCA) units. A more current type B strain replaces that in the 1971-72 formulation. Each adult dose of 1972-73 vaccine contains a total of 700 CCA units type A [A Aichi/2/68(H3N2)] ** and 300 CCA units type B (B Massachusetts/1/71). Doses for children are specified in the manufacturers' package labeling. Vaccines from all producers are of the highly purified variety and should be less often associated with adverse reactions than the previous influenza vaccines.

VACCINE USAGE

General Recommendations

Annual vaccination is recommended for persons of all ages who have chronic debilitating conditions: 1) congenital and rheumatic heart disease, especially with mitral stenosis, and arteriosclerotic and hypertensive heart disease, particularly with cardiac insufficiency; 2) chronic bronchopulmonary diseases, such as asthma, chronic bronchitis, cystic fibrosis, bronchiectasis, emphysema, and advanced tuberculosis; 3) diabetes mellitus and other chronic metabolic disorders.

Although the value of routinely immunizing all older age persons is less clear, those patients who have incipient or potentially chronic disease, particularly affecting cardiovascular and bronchopulmonary systems, should also be considered for annual immunization.

Immunizations of persons who provide essential community services may also be considered if local priorities justify. However, before undertaking such programs, responsible physicians must take into account a number of reasonable constraints: difficulties inherent in predicting influenza epidemics, variability of vaccine effectiveness, incidence of adverse side effects, cost, availability of vaccine, and risk of diverting vaccine from those with chronic debilitating conditions who are at risk.

Schedule

The primary series consists of 2 doses administered subcutaneously, preferably 6-8 weeks apart. (Dose volume for adults and a detailed schedule for children are specified in the manufacturers' labeling.) Persons who have had 1 or more doses of vaccine containing the Hong Kong strain antigen (all influenza vaccines since 1968-69) need only a single subcutaneous booster dose of bivalent vaccine. All others should receive the full primary series. Vaccination should be scheduled for completion by mid-November.

Precautions

Influenza vaccine is prepared from viruses grown in embryonated eggs and ordinarily should not be administered to persons clearly hypersensitive to egg protein, ingested or injected.

*Official name: Influenza Virus Vaccine, Bivalent.

**The World Health Organization has recommended a revised system of nomenclature for type A influenzaviruses which includes their strain designation and a description of the two surface antigens, hemagglutinin (H) and neuraminidase (N).

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RECOMMENDATION OF THE PUBLIC HEALTH SERVICE ADVISORY COMMITTEE ON IMMUNIZATION PRACTICES

INFLUENZA VACCINE: Supplementary Statement

Preliminary information from various international sources and from the World Health Organization's Influenza Centers indicates that in recent months, moderately distinctive but not altogether new strains of type A influenzavirus have caused outbreaks of illness in Southeast Asia, Australia, and the Far East and a few cases in Hawaii. Cases have been clinically characteristic of influenza.

The newer influenzaviruses are closely related to each other and to strains identified in southern India, July 1971, and in England, January 1972. The prototype antigen in widest use for comparative laboratory tests of current strains is A/England/42/72(H3N2).

Surveillance will be intensified to determine the extent of spread of the newer strains. It is reasonable to expect that they will be the cause of influenza cases in the United States during the 1972–73 season. However, it cannot now be determined whether widespread outbreaks are likely to occur. This is partly because a majority of our population already has some immunity from prior exposure to related influenzaviruses.

Based on laboratory information, currently available influenza vaccine should offer some protection against the newer strains. Its use for high risk groups (MMWR, Vol. 21, No. 24) is strongly recommended. The dosage and scheduling previously stated still apply.

STATE EPIDEMIOLOGISTS

Key to all disease surveillance activities are those in each State who serve the function as State epidemiologists. Responsible for the collection, interpretation and transmission of data and epidemiologic information from their individual States, the State epidemiologists perform a most vital role. Their major contributions to the evolution of this report are gratefully acknowledged.

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